Intracranial Angiography in the Treatment of Cerebral Vascular Accidents

Reports of Cases and a Review of the Literature

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SUMMARY

Five cases of intracerebral hematoma, including one case of calcified intracerebral hematoma, are presented and treatment is discussed.

The role of carotid arteriography, stellate ganglion blocks, vasodilator drugs and operation in the treatment of cerebral vascular accidents is discussed and typical cases of the various vascular anomalies of the cerebral vascular system are presented.

THE successful surgical treatment of a number of cerebral vascular lesions including five cases of intracerebral hematoma prompted a review of the literature concerning the methods of treatment of lesions of this type.

During the past decade or so there has been a change in the therapeutic approach to the problem of intracranial vascular accidents. Penfield[38] in 1933 said, "The right of the aged and hypertensive to die from apoplectic stroke undisturbed has been unchallenged for a long time." He concluded, after reviewing records of patients observed by him, that with careful study and selection of patients it "might be possible to do something for a larger number of them." Cushing,[14] Craig and Adson,[12] Rankin,[41] Pilcher,[39] Woodhall,[58] Klemme,[27] Karlen,[26] King[29] and Abbott[1] are a few among the many who have successfully challenged the right of these patients to "die undisturbed." Huesner[24] in 1888 was probably the first surgeon to report the evacuation of an intracerebral hematoma with recovery of the patient. In 1941 Meridith[36] reported that only 17 cases of surgically treated intracerebral hematoma were reported in the medical literature. By 1943 that number had increased to 33. Recently many instances of successful surgical treatment of cerebral vascular lesions of the type hitherto regarded as hopeless have been reported in the literature. These include the surgical removal of intraventricular hematomas,[13] ligation of aneurysms of the left middle cerebral arteries[49] and the successful treatment of lesions of the basilar and vertebral arteries.[30] Dandy[15] stated that he knew of no successful attack on lesions of those vessels, and his report of a sudden death upon manipulation of one vertebral artery has made most surgeons loath to work in that area.

With the more general use of the carotid arteriogram as an adjunct in the diagnosis, the indications for surgical aid for the patient with a cerebral vascular lesion are more definite. When the arteriogram is done by the percutaneous method as first demonstrated by Lowman and Meyerson[35] in 1936 and later by Shmidzu,[48] Brobeil,[7, 8] Kristiansen[31] and Lindgren,[33] it spares the critically ill patient the minor trauma incident to exposing the carotid artery in the neck. King,[28] Engeset,[16] Radner[40] and others have described similar methods for injecting the vertebral arteries. Wechsler,[57] List[34] and Gariepy,[17] to mention a few among many, have stressed the fact that carotid angiography is a safe diagnostic procedure that does not increase the surgical risk for patients who are in a precarious condition. List[34] stated, "It [angiography] carries little risk even in patients with high intracranial pressure since it does not interfere with intracranial hydrodynamics." A consideration of the mechanisms involved makes it readily apparent that the force that can be exerted through a 19-gauge needle by hand pressure on a rapidly flowing column of blood, in a vessel as large as the common carotid artery, is negligible; especially when it is remembered that the total flow of blood through the brain in the human is in excess of 600 cc. per minute.[19, 46]

According to Aring and Merritt,[4] Albertini,[2] Taylor and Page,[53] Buzzard,[9] Bidwell, [5] Church[10] and others, clinical differentiation of hemorrhage, thrombosis and embolism is not difficult. After studying two series of patients totaling 652 cases, Aring and Merritt concluded, "A thorough study of the history with the results of the physical and neurologic examinations and examination of the spinal fluid should make a differentiation possible in nearly 100 per cent of the cases." They pointed out that sudden severe headache, vomiting, stiffness of the neck and progression of symptoms are strongly indicative of hemorrhage and that these findings were present in 51 per cent of the hemorrhage cases reviewed. Spinal fluid pressure greater than 300 mm. of water did not occur with thrombosis but was noted in 38 per cent of the cases of hemorrhage. In 18 per cent of the patients with hemorrhage the spinal fluid pressure was above 400 mm. of water. Blood in the spinal fluid was observed macroscopically in 74 per cent of the patients with hemorrhage and in only 0.15 per cent of those with thrombosis.

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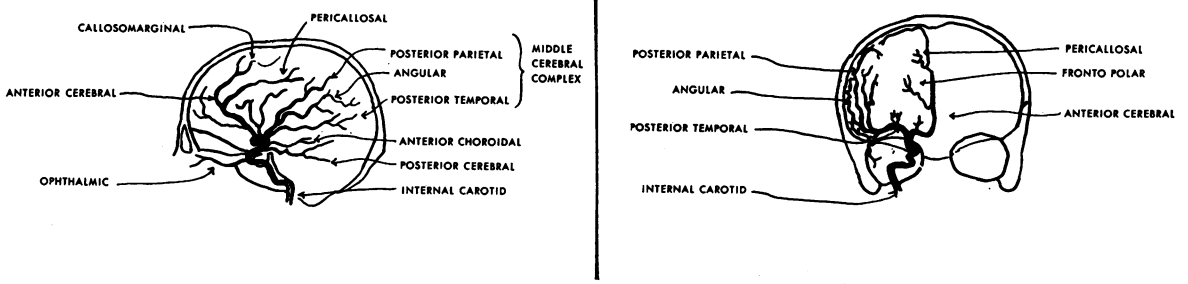
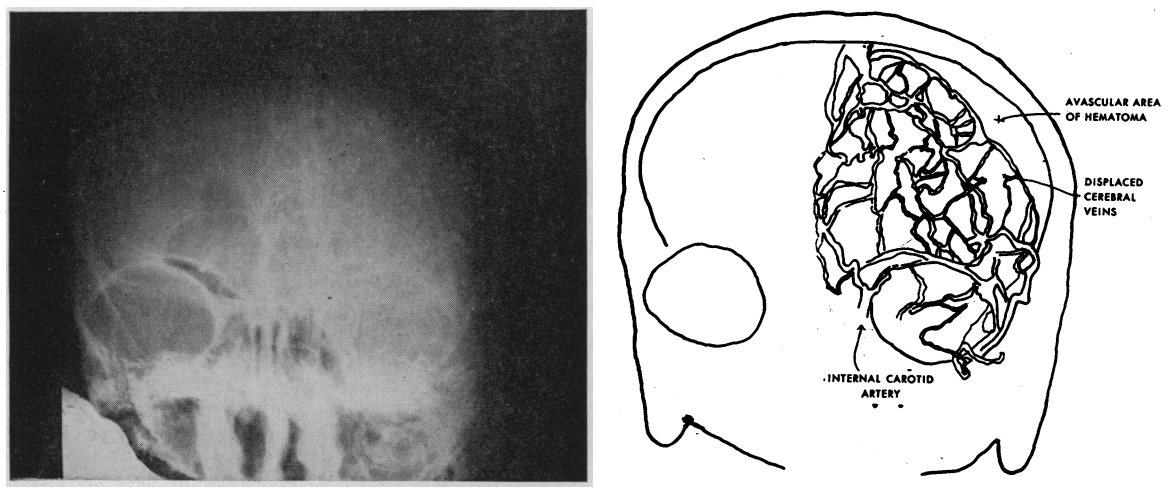
Rowbotham[44] said that in cases of intracerebral bleeding the spinal fluid contains blood. The authors did not find this to be so in cases observed by them (Case 4, intracerebral hematoma; and Case 8, traumatic arteriovenous fistula).

The frequency of occurrence of the various types of lesions in a series of 407 cases studied at the Boston City Hospital was: Thrombosis, 81.15 per cent; hemorrhage, 15.0 per cent; embolism, 3.2 per cent.

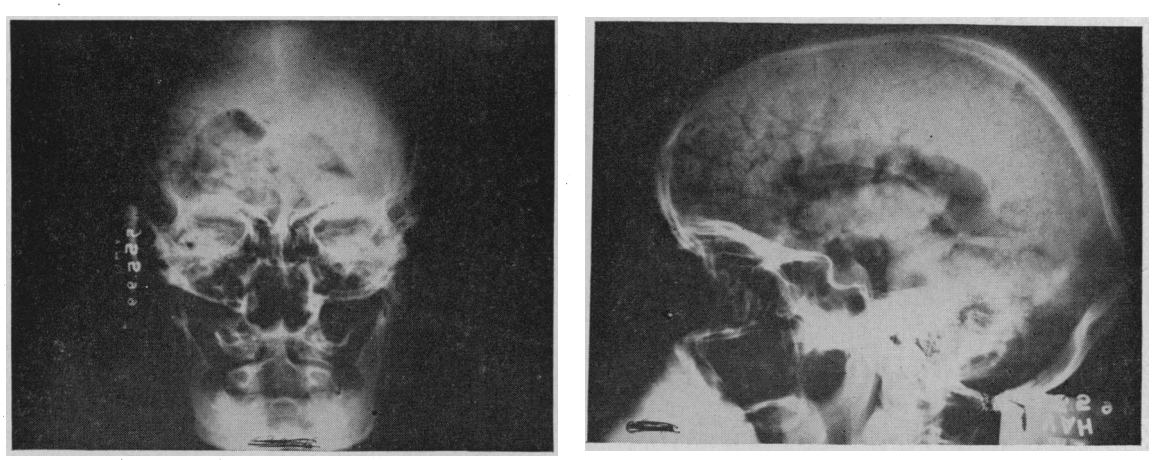
Alvarez[3] pointed out the necessity of taking a



A. Tracings of normal arteriograms.



B. Angiogram and tracing of a subdural hematoma.



C. Pneumoencephalogram showing depression of left lateral ventricle by subdural hematoma.

Figure 1

careful history from older people, in whom, he believes, small, usually unrecognized strokes are a common cause of illness. Globus[18] stressed that there is, in many patients, ample evidence of the disease process prior to the first episode of hemorrhage.

Where clinical signs are not of a localizing nature, as is often the case, Woodhall[58] and others report successful localization of the hemorrhage by electroencephalogram. This method has been of aid to the authors in several cases. Groff and Grant[21] pointed out that the clinical picture occurring with rupture of the middle meningeal artery or one of its main branches is often not sufficiently characteristic to differentiate it from other intracranial hemorrhages, but that the angiogram is strikingly characteristic. This is well illustrated in one of the cases observed by the authors (Figure 1, *b* and *c*). Shenken[47] stated that in cases of rupture of the middle meningeal artery or one of its main branches, surgical treatment, to be of any value, must be given within three to six hours or before decerebration occurs.

In cases of thrombosis or cerebral embolism, pronounced improvement in the clinical picture is often obtained by the use of stellate ganglion block and by the use of vasodilating drugs. De Takats,[52] Volpitto,[56] Risteen,[43] Gariepy[17] and others have reported rather amazing results following the use of stellate ganglion block. The authors have observed cases in which aphasia, hemiparesis and lesser neurologic disorders disappeared after stellate ganglion block, often done bilaterally. Rankin,[41] Cobb,[11] Ochsner and DeBakey[37] and others have pointed out the importance of relieving the vasospasm incident to cerebral thrombosis and embolism. Risteen and Volpitto[43] observed, during operation, the vasodilating effects of stellate ganglion block on the cerebral vessels. Villeret and Cachera,[55] working with experimental animals, observed through a window in the skull that, in all cerebrovascular accidents, surrounding the hemorrhagic or ischemic zone there is a zone of stasis and vasoparalysis with segmental spasm of the vessels. De Takats and Gilbert[52] recommended the use of aminophylline in doses of 0.3 to 0.5 gm. given intravenously with stellate block. They noted in animals that bilateral stellate block was much more effective than unilateral block. Reese[42] reported on the beneficial effects of 0.12 gm. of aminophylline thrice daily in arteriosclerotic patients with neurologic deficits. Howard[25] and coworkers, who did extensive work on the physiologic action of aminophylline, reported that it reduced venous pressure, stimulated respiration and shortened circulation time.

Intracerebral Hematoma:

CASE REPORTS

CASE 1: A 30-year-old white male, admitted to the hospital in a comatose state, had complained of headache that morning and at noon was found unconscious. There was nothing of significance in the past history.

At the time of examination the patient was unconscious, lying with the left arm and leg flexed. There were convulsive motions of the right arm. The skin temperature was definitely lowered on the entire right side of the body. The pupils were equal and reacted to light. The neck was very stiff and Brudzinski and Kernig signs were present. Reflexes were increased on the right and Hoffmann's sign on the right was noted. Response to painful stimuli was greater on the left side of the body than on the right. There was gross blood in the spinal fluid and the pressure was 490 mm. of water.

The patient remained comatose. The spinal fluid gradually cleared to a xanthochromic color. A left common carotid arteriogram on the eighth day (Figure 2, *a* and *b*) showed posterior-inferior displacement of the middle cerebral complex of vessels and anterior-inferior displacement of the anterior cerebral artery.

A ventriculogram (Figure 2, *c*) revealed depression of the frontal horn of the left lateral ventricle. A 6-cm. intracerebral clot was removed from the left frontal lobe, leaving a smooth-walled cavity without bleeding points. The postoperative course was uneventful and the neurologic deficits disappeared. The patient returned to work after a convalescence of six weeks, and a three-year follow-up was uneventful.

CASE 2: A 33-year-old white female was admitted to the Sonoma County Hospital with a purulent discharge from the right ear. Subtotal mastoidectomy had been done 21 days before, and three days later the patient complained of headache, became very talkative in a repetitious manner and showed pronounced loss of normal inhibitions. This state was followed by profound lethargy from which the patient could be aroused only with great difficulty. The following day the pupil of the right eye dilated, and then both pupils became fixed to light. However, slow improvement followed, and upon spinal fluid tap the pressure was normal and the fluid xanthochromic. There were 22 lymphocytes present in each cubic mm. The patient was discharged from the hospital and was readmitted seven days later with the previously noted complaints.

The patient was semi-comatose. The blood pressure was 130 mm. of mercury systolic and 80 mm. diastolic, and the temperature was 98.4° F. The pupils were equal and reacted to light. Pronounced stiffness of the neck was noted. Papilledema was not present. The patient did not move the left extremities. The reflexes were increased on the left and response to painful stimuli applied to the left side of the body was less than to the same stimuli applied on the right. A right homonymous hemianopsia had been detected before the patient became comatose. Pronounced xanthochromia was noted in the spinal fluid. Spinal fluid pressure was 290 mm. of water and there were 25 lymphocytes present in each cubic mm. The protein content was 102 mg. per 100 cc.

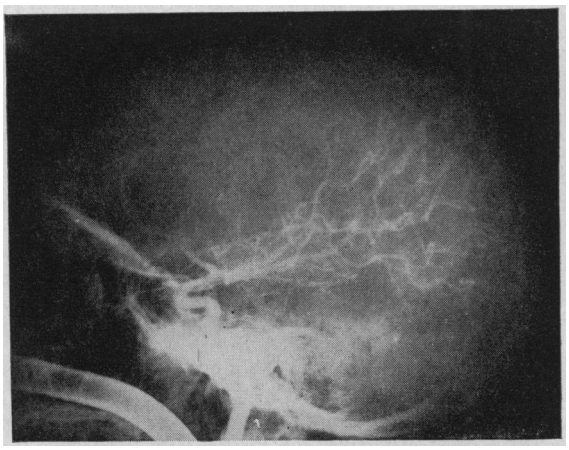
On the basis of these findings a burr hole was made over the anterior part of the right temporal lobe and an intracerebral hematoma containing 90 cc. of old blood was evacuated, leaving a cavity lined with old blood pigment which was washed out.

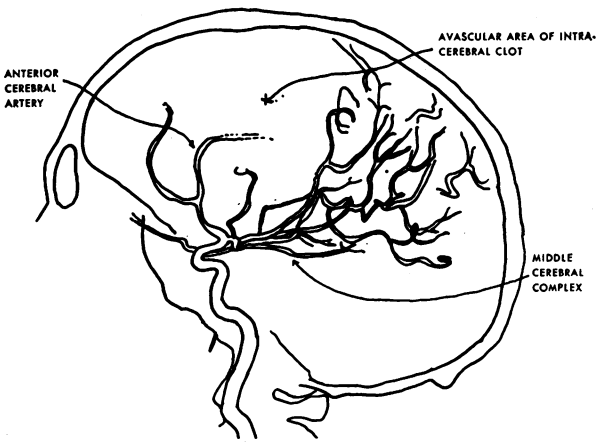
The patient regained consciousness on the operating table and the postoperative course was uneventful for the follow-up period of 18 months.

CASE 3: A 52-year-old white male was admitted with complaints of intense occipital headache, nausea and vomiting. There was weakness of the left side of the body.

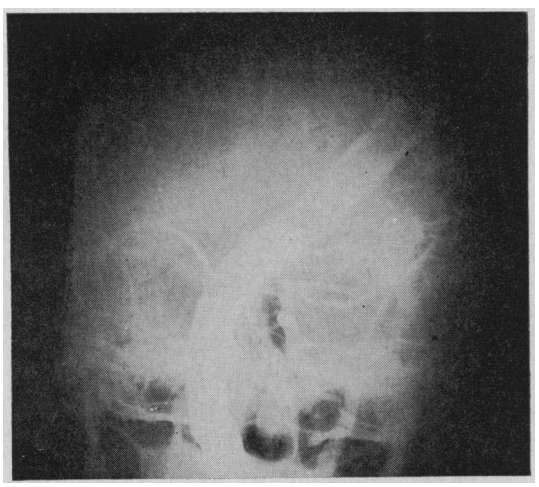
There was a history of hypertension for many years. The severe occipital headaches had been present for four months. Four days before admission the patient had awakened with headache and soreness and stiffness of the neck, and had vomited.

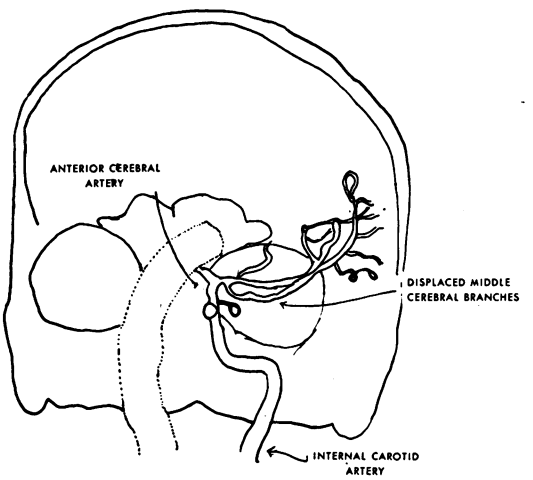
The patient appeared to be confused. The blood pressure was 160 mm. of mercury systolic and 90 mm. diastolic. The



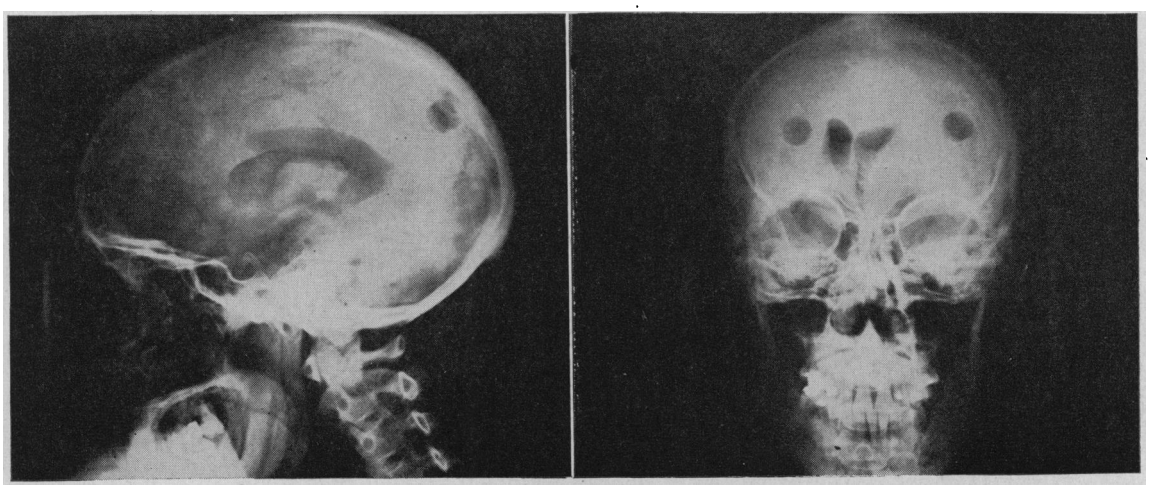


A. Intracortical clot. Angiogram showing displacement of the middle and anterior cerebral arteries.





B. Intracortical clot. Anterior-posterior angiogram.



C. Ventriculogram of above case – depression and shift to right of left lateral ventricle.

Figure 2

pupils were equal in size and responded to light. There was one diopter of papilledema bilaterally. The neck was very stiff. Spastic left hemiplegia and a left hemihypalgesia were noted. Hoffmann's sign and Babinski's sign were present on the left side. The spinal fluid was xanthochromic and at a pressure of 260 mm. of water. A right common carotid arteriogram was done and an abnormal vascular pattern was apparent in the distribution of the right middle cerebral artery.

A ventriculogram revealed depression of the right lateral ventricle with medial shift of the anterior horn. A semiliquid intracerebral hematoma was evacuated from the right frontal lobe. Recovery from the hemiplegia was uneventful and sensory deficit cleared. The patient remained well during a two-year follow-up.

CASE 4: A 23-year-old white male was admitted to the Merced County Hospital with complaint of weakness and numbness of the left extremities and inability to talk.

Well until the day of entry, the patient had noticed while at school that he could not hold a pencil in the left hand. Three hours later he noticed weakness in the left leg and he became aphasic. In x-ray films of the head enostosis of the parietal skull on the right was noted, and intracerebral calcifications were observed beneath the enostosis. A presumptive diagnosis of meningioma was made.

The patient was lethargic with spastic left hemiparesis and left hemihypesthesia. He was nauseated and vomiting. The left tendon reflexes were increased but no pathologically significant signs in fingers or toes were present. There was complete astereognosis in the left hand. The spinal fluid was normal. A small firm tumor of the right parietal scalp, presumably a lipoma, was noted.

The lethargic state progressed to coma, with severe headache and vomiting. Papilledema developed. A right carotid arteriogram revealed a large group of abnormal venous channels in the right parietal area.

A ventriculogram revealed displacement of the right lateral ventricle consistent with a high parietal mass on the right. At operation a number of abnormally large veins were encountered on the surface of the cortex. A large intracerebral clot was evacuated from the right parietal lobe. It contained calcium with old organized clot and fresh clot of jelly-like consistency. The patient gradually recovered from the paresis and the speech defect and remained well during a two-year follow-up.

CASE 5: A 27-year-old white male was admitted with complaints of left-sided headache, nausea, vomiting, blurred vision and transient attacks of complete aphasia.

There was history of a right-sided convulsion with unconsciousness at six months of age, with similar attacks intermittently for the ensuing six months. Three years prior to admission the patient had received a blow to the left side of the head in an auto accident. This had been followed by weakness of the right arm and leg, nausea and vomiting.

Hemiatrophy of the right side of the body was noted upon physical examination. The pupils were equal and reacted to light. There was no papilledema. There was right hemiparesis and hemihypesthesia with increased reflexes, but no pathologically significant signs in fingers or toes. Partial motor aphasia was noted. The spinal fluid was clear and the pressure was normal. A left common carotid arteriogram revealed a mass in the left parietal lobe.

A large mass of clotted thin-walled veins and an old organized hematoma were removed from the left parietal lobe. Postoperatively, mild motor and sensory loss on the right side of the body persisted. During a two-year follow-up the patient had one transient attack of motor aphasia.

Aneurysm of the Middle Cerebral Artery:

CASE 6: A confused, disoriented, 51-year-old white male was admitted with complaints of severe bitemporal headache, nausea, vomiting and stiffness of the neck.

The patient had been well until four days before admission, when severe bitemporal headache and nausea suddenly developed and coma lasting 20 minutes followed.

The pupil of the left eye was large and reacted sluggishly to light. There was left spastic hemiparesis without pathological reflexes. The spinal fluid was xanthochromic and the pressure was 410 mm. of water. A right common carotid arteriogram revealed an aneurysm of the middle cerebral artery (Figure 3, *a*). The patient died before he could be prepared for operation. Hemorrhage from aneurysm was demonstrated at autopsy.

Thrombosis of the Middle Cerebral Artery:

CASE 7: A 22-year-old Japanese male was admitted with spastic right hemiplegia and partial motor aphasia.

Five years previously, weakness of the right arm and aphasia had developed suddenly and a generalized convulsion had followed. The aphasia and weakness gradually cleared over a three-year period. A year previously the weakness and aphasia recurred and persisted for three weeks. The patient was hospitalized at that time and a diagnosis of multiple sclerosis was made. The condition remained unchanged.

Upon examination, partial motor aphasia, alexia and atrophy of the right extremities, superior right quadrantic hemianopsia, right hemiplegia with flaccidity, and right hemihypesthesia were noted. Hoffmann's and Rossolimo's signs were present. The spinal fluid was normal. A left internal carotid arteriogram revealed thrombosis of the left middle cerebral artery.

Bilateral stellate ganglion blocks and aminophylline gave only temporary improvement, probably due to the age of the lesion (five years).

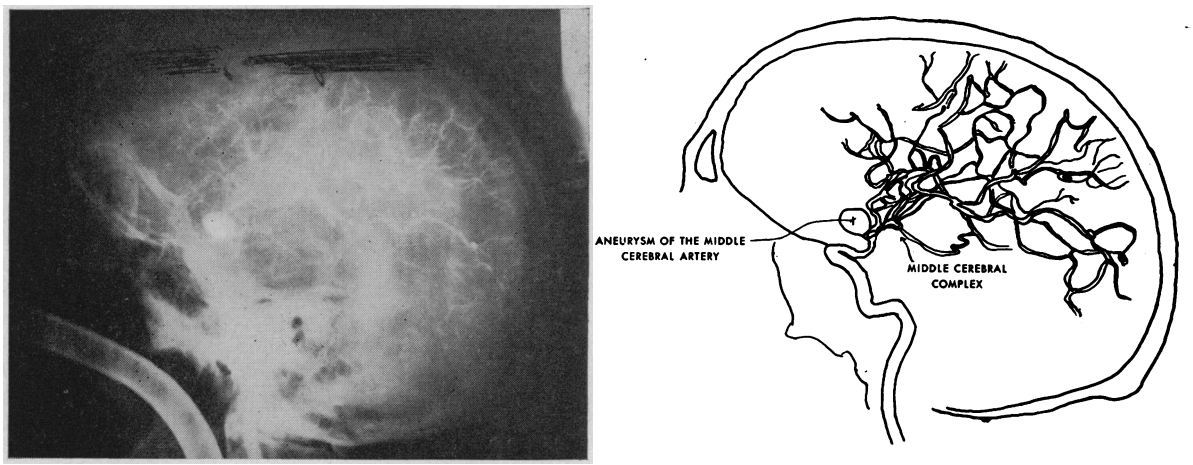
Traumatic Arteriovenous Fistula:

CASE 8: A 30-year-old white male was admitted with a painful pulsating exophthalmus of the left eye, nausea, headache and blurring of vision.

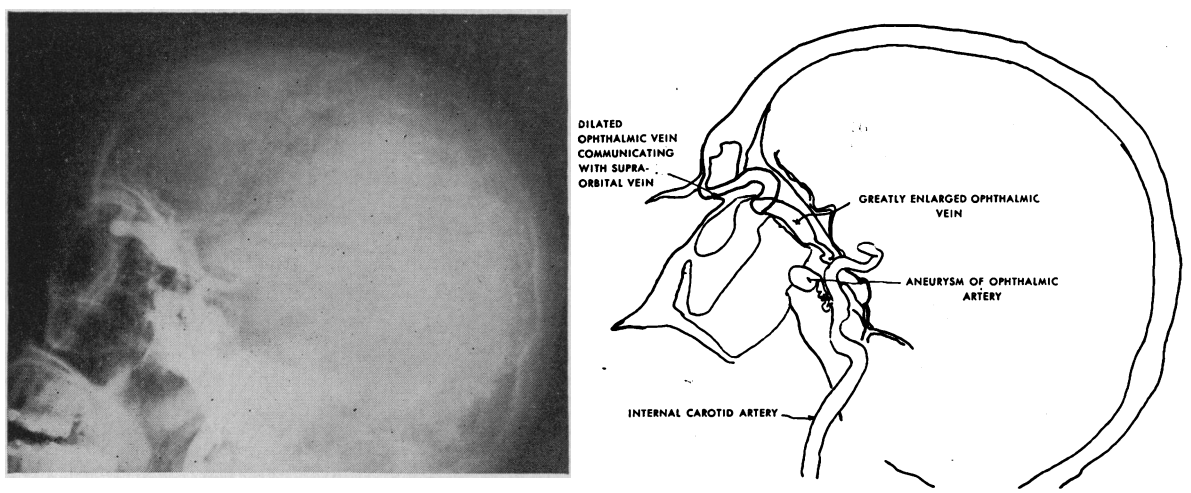
Two months previously the patient had received fractures of the left ankle and of the left ramus of the mandible, and abrasions of the left forehead, in an airplane crash. Three weeks before admission, pain in the left eye developed and was followed shortly by protrusion of the left eye and a roaring in the head.

Upon examination, pronounced proptosis of the left eye and moderate periorbital edema were noted. A loud systolic bruit was heard on auscultation of the head. Occlusion of the left carotid artery decreased the bruit. There was paralysis of all the external ocular muscles of the left eye and the pupil was fixed in the mid-position, indicating autonomic paresis as well as third, fourth and sixth nerve paralysis. There was loss of sensation over the first and second divisions of the trigeminal nerve on the left. A left carotid arteriogram (Figure 3, *b*) confirmed the clinical impression of a lesion of the left cavernous sinus.

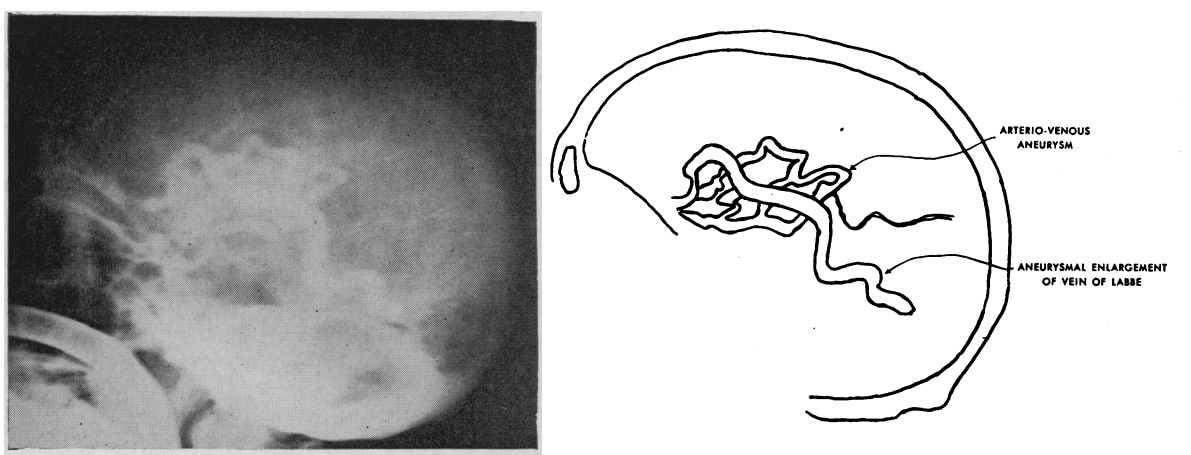
Intermittent occlusion of the left carotid artery was carried out. A clamp was devised which permitted occlusion to the point that the noise in the head could not be heard. The period of occlusion was gradually increased until it could be maintained for 60 minutes. The left common carotid artery was then ligated. This decreased the proptosis, headache and bruit. Ten days later the left external and internal carotid arteries were ligated. This resulted in disappearance of the bruit and gradual reduction of proptosis. During the two-and-a-half-year follow-up proptosis disappeared entirely and there was no recurrence of symptoms.



A. Aneurysm, left middle cerebral artery — lateral view.



B. Arterio-venous fistula, right carotid and cavernous sinus.



C. Arterio-venous aneurysm supplied by middle cerebral artery.

Figure 3

Arteriovenous Aneurysm:

CASE 9: A 29-year-old white male was admitted with complaints of inability to talk, double vision and dizziness, with blurring of vision on turning the head to the left.

Twelve months previously, motor aphasia developed suddenly and was followed by gradual development of blurring of vision, nausea and coma. Spinal fluid pressure at that time was 310 mm. of water and the fluid was xanthochromic. At that time the patient was found to have bilateral papilledema and anopsia in the right lower quadrant. These conditions gradually cleared over a period of several months. Then aphasia and blurring of vision gradually returned, beginning about a month prior to the present admission.

The pupil of the right eye was large. Bilateral papilledema and right lower hemianopsia were noted. There was partial motor aphasia and right hemiparesis with increased reflexes. Stereognosis was impaired on the right. The spinal fluid was clear and the pressure was 250 mm. of water. A left carotid arteriogram (Figure 3, *c*) revealed a mass of enlarged vessels having the appearance of a cavernous hemangioma. The patient was not willing to undergo operation and was given a course of deep x-ray therapy which did not alter the clinical condition.

Arteriovenous Aneurysm of the Scalp:

CASE 10: A 26-year-old white male was admitted with complaints of pain over the left side of the head, and in the left eye, and a buzzing noise in the left side of the head.

Nine years previously the patient had received a severe blow to the head in an automobile accident. A soft swelling over the left temple had persisted since the time of the accident. The noise and the pain had been first noticed two years previously.

Upon examination, a soft, smooth, non-pulsating swelling over the left side of the head was observed. There was a thrill on palpation, and upon auscultation a high-pitched bruit, which disappeared on compression of the left carotid artery, was noted. There were no other significant neurologic findings. A large "port wine stain" of the left cubital fossa was noted. A phonocardiogram revealed that the bruit had both systolic and diastolic components. A left common carotid arteriogram revealed an arteriovenous angioma of the superficial, temporal and frontal arteries of the scalp. This lesion was removed completely with immediate relief of signs and symptoms. The patient remained well during a three-year follow-up.

DISCUSSION

It is of interest to note that in several of the cases of intracerebral hematoma here reported, a smooth-walled cavity remained after removal of the clot. This finding is in agreement with the observations of Scheinker,[45] Craig and Adson[12] and many others who have expressed belief that many spontaneous intracerebral hemorrhages are of venous origin. Sachs, in discussing this problem,[45] pointed out that in children spontaneous intracerebral hemorrhages are almost always of venous origin. In one patient treated by the authors (Case 5) the hemorrhage was undoubtedly of venous origin, as many of the thin-walled veins were observed in the clot on histologic examination.

The presence of calcium in an intracerebral hematoma (Case 4) has been previously observed.[32] Grantham and Smolik[20] stated in 1942 that only two such cases were reported in the medical literature.

Hamby[23] and Browder and Turney,[6] in discussing the disposition of the intracerebral blood in hemorrhage, reported that in necropsy of patients who died a few hours after the hemorrhage occurred, clotted blood was found, but that in those who survived 12 to 15 hours the intracerebral blood had become syrupy and suitable for aspiration.

As to the indications for surgical intervention in cases of cerebral vascular accidents, Gurdjian[22] observed: "In acute hemorrhagic conditions, the one important symptom justifying exploration is increasing drowsiness or stupor." Craig and Lipscomb[13] stated that in all cases of expanding lesions of the brain, operation should be considered not only to relieve the symptoms but to establish a diagnosis. Meridith[36] expressed the opinion that "No patient, however moribund, should be denied the benefit of at least a limited procedure—as often a surprisingly happy solution will be encountered which is undoubtedly life saving to the patient and of considerable satisfaction to all those concerned with the case." Hamby[23] expressed belief that operation should be considered for patients with apoplectic or with gradual onset of symptoms, with progression of the signs and symptoms and development of signs of increasing intracranial pressure. Penfield,[38] in summarizing a report of cases observed by him, felt that had he been more prompt in giving surgical aid, the life of one patient could have been saved and residual hemiparesis that occurred in a 14-year-old boy could have been prevented.

The authors believe that at least an angiogram and at least a limited surgical procedure as a step toward diagnosis and a more positive approach toward the saving of life is warranted for patients who survive the shock of hemorrhage yet show no evidence of improvement.

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